



**PROGRAM
of the**

***One Hundred Thirty-First
Annual Meeting***

**AMERICAN
OTOLOGICAL SOCIETY, INC.**

**May 9-10,
1998**

**The Breakers
Palm Beach, Florida**

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JULY 1, 1997- JUNE 30, 1998

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The American Otological Society is accredited by the
Accreditation Council for Continuing Medical Education
to sponsor continuing medical education for physicians.

**This Continuing Medical Education offering meets the criteria for eight (8)
credit hours in Category One (1) of the Physician's Recognition Award of
the American Medical Association.**

SATURDAY, MAY 9, 1998

REGISTRATION - 12 Noon

BUSINESS MEETING - 12:30 p.m.

**ROOM: MEDITERRANEAN BALLROOM
(Restricted to Members)**

Minutes of the Annual Meeting 1997

Introduction of New Members

Election of Nominating Committee

Report of the Secretary-Treasurer

Report of the Editor-Librarian

SCIENTIFIC PROGRAM - 1:00 p.m.

**ROOM: MEDITERRANEAN BALLROOM
(Open to Non-Members)**

Remarks by the President

Charles M. Luetje, MD

Remarks by the Guest of Honor

Robert A. Jahrsdoerfer, MD

Presidential Citation

Jack V. D. Hough, MD

Special Presidential Awards

Howard P. House, MD

George E. Shambaugh, Jr., MD

Notes

Stapes Surgery

1. 1:20 p.m. Hearing after Laser Stapedotomy with Preservation of the Stapedius Tendon
Herbert Silverstein, MD*
Seth L. Rosenberg, MD
T. Oma Hester, MD
 2. 1:28 p.m. Anesthesia for Stapedectomy
Jack J. Wazen, MD*
Beth Wambach, MD
Arlene Markowitz, MD
 3. 1:36 p.m. Experience with Stapes Surgery in a Large Teaching Institution; the Role of the Staff Supervising Surgeon in Outcomes
Peter C. Bondy, MD*
Lorenz F. Lassen, MD, FACS
 4. 1:44 p.m. Stapedectomy for Far-Advanced Otosclerosis
Paul F. Shea, MD*
Xianxi Ge, MD
John J. Shea, Jr., MD
- 1:52 p.m. **Discussion**

Chronic Ear Surgery

5. 2:10 p.m. The Effect of Gelfilm in the Prevention of Fibrosis in the Animal Model
Michael A. McGhee, MD*
John L. Dornhoffer, MD

***speaker**

Notes

6. 2:18 p.m. Epitympanic Approach Towards
Cholesteatoma
John L. Dornhoffer, MD, FACS

7. 2:26 p.m. Supralabyrinthine Approach to the
Petrous Apex
Fred F. Telischi, MD*
Michal Luntz, MD

8. 2:34 p.m. Long-Term Middle Ear Ventilation
During Tympanoplasty with a Sub-
annular T-Tube
Timothy O'Hare, MD, PhD*
Joel A. Goebel, MD, FACS

2:42 p.m. **Discussion**

2:50 p.m. **Break**

Sensorineural Hearing Loss

9. 3:20 p.m. ABR Hearing Screening for High-Risk
Infants
Paul R. Kileny, PhD*
Lori A. Van Riper, MS

***speaker**

Notes

10. 3:28 p.m. **Sensorineural Hearing Loss Following Occlusion of the Enlarged Vestibular Aqueduct**
D. Bradley Welling, MD
Patrick W. Slater, II, MD*
Michael D. Martyn, MD
Bruce J. Gantz, MD
William M. Luxford, MD
Clough Shelton, MD
11. 3:36 p.m. **Influence of Mitochondrial Metabolite Supplements on Age-Related Hearing Loss**
Michael D. Seidman, MD
Mumtaz J. Khan, MD*
Uma Bai, PhD
Najeeb Sherwany, MD
12. 3:44 p.m. **Progressive Sensorineural Hearing Loss, Subjective Tinnitus and Vertigo Caused by Diabetes Mellitus**
Jack L. Pulec, MD*
Marlene B. Pulec
Ignacio Mendoza H., MD

3:52 p.m. **Discussion**

***speaker**

Light Microscopy and Ultrastructure

13. 4:00 p.m. **Histologic Changes of the Cochlea
After Automobile Air Bag Deployment**
Douglas E. Mattox, MD*
Weihua Lou, MD
Joel Kalb, PhD
C. Richard Price, PhD
14. 4:08 p.m. **Does Otosclerosis Occur Only in the
Temporal Bone?**
Pa-Chun Wang, MD
Saumil N. Merchant, MD*
Michael J. McKenna, MD
Robert S. Glynn, ScD
Joseph B. Nadol, Jr., MD
15. 4:16 p.m. **The Otological Aspects of Usher's
Syndrome: Classification, Histo-
pathology and Management**
Arvind Kumar, MD, FRCS (Edin)*
Isamu Sando, MD
Haruo Takahashi, MD
Gerald Fishman, MD
Reena Dhanda, BA
16. 4:24 p.m. **Construction and Characterization of a
Human Acoustic Neuroma (Vestibular
Schwannoma) cDNA Library**
Phillip A. Wackym, MD*
Elizabeth Toh, MD
Marta Troyanovskaya, PhD

***speaker**

Notes

17. 4:32 p.m. Electron Microscopic Study on Cystic Vestibular Schwannoma
Jens Thomsen, MD, DMSc, FRCPS*
Samih Charabi, MD, DMSc
Klaus Qvortrop, MD, PhD
Mirko Tos, MD, DMSc

4:40 p.m. **Discussion**

Vertigo

18. 4:48 p.m. Paroxysmal Positional Vertigo Syndrome
Vincente Honrubia, MD, DMSc*
Marjorie R. Harris, MA
Robert W. Baloh, MD

19. 4:56 p.m. Paroxysmal Positional Vertigo: Idiopathic vs. Post-Traumatic
Athanasios Katsarkas, MD, MSc*

5:04 p.m. **Discussion**

5:15 p.m. **GROUP PHOTOGRAPH**
MEMBERS OF THE AMERICAN
OTOLOGICAL SOCIETY, INC.
(Location to be announced)

***speaker**

Notes

SUNDAY, MAY 10, 1998

REGISTRATION - 7:00 a.m.

BUSINESS MEETING - 7:00 a.m.

ROOM: MEDITERRANEAN BALLROOM
(Restricted to Members)

REPORTS OF THE:

- A. Board of Trustees of the Research Fund
- B. American Board of Otolaryngology
- C. Award of Merit Committee
- D. American College of Surgeons
- E. American Academy of Otolaryngology
Head and Neck Surgery

Report of the Audit Committee

Report of the Nominating Committee

Report of Communications

Unfinished Business

New Business

SCIENTIFIC PROGRAM -7:45 a.m.

ROOM: MEDITERRANEAN BALLROOM
(Open to Non-Members)

Opening Remarks

Charles M. Luetje, MD

Presentation of the Life Achievement
Award (On behalf of the American
Auditory Society)

Richard T. Miyamoto, MD

Howard P. House, MD

Introduction of New Director, NIDCD

James Battey, MD, PhD

Notes

Cochlear Implantation

20. 8:00 a.m. **Mastoidotomy Tympanotomy Approach for Cochlear Implantation - Advantages of this Technique. A Multicenter Multi-national Study**
Marcos V. Goycoolea, MD, PhD*
Hamlet Suarez, MD
Santiago Arauz, MD
Gloria L. Ribalta, MD
21. 8:08 a.m. **Deep Insertion of Cochlear Implant Electrodes**
Thomas Balkany, MD, FACS*
Eloy Villasuso, MSIV
Annelle V. Hodges, PhD
Phillip A. Bird, FRACS
22. 8:16 a.m. **Multichannel Cochlear Implantation in Children with Cochlear Ossification**
Ronald L. Steenerson, MD*
Lucinda B. Gary, MA
23. 8:24 a.m. **Management of Cochlear Implant Infections**
Jay T. Rubinstein, MD, PhD*
Bruce J. Gantz, MD
Wendy S. Parkinson, MA
24. 8:32 a.m. **The Nucleus 24 System in Children**
Noel L. Cohen, MD*
Susan B. Waltzman, PhD
Steven J. Staller, PhD
- 8:40-8:55 a.m. **Focused discussion of preceding papers**

***speaker**

Notes

25. 8:56 a.m. Comparison of Audiologic Performance Following Reimplantation: A Multi-center Overview
AnnMarie Henson, MEd, CCA-A*
William H. Slattery, III, MD
Dawna Mills, MA, CCC-A
26. 9:04 a.m. Cochlear Implant MRI Compatibility
Noel L. Cohen, MD
J. Thomas Roland, Jr., MD*
27. 9:12 a.m. Positron Emission Tomography (PET) in Cochlear Implant Recipients
Richard T. Miyamoto, MD*
Donald Wong, PhD
David B. Pisoni, PhD
Gary Hutchins, PhD
Mark Sehgal, MD
28. 9:20 a.m. Variations in Central Nervous System Activation Between Cochlear Implant Users Receiving Maximal or Minimal Benefit
Peter S. Roland, MD*
Mike S. Devous, PhD
Emily A. Tobey, PhD
Jay S. Perrin, MS
Kelley Payne, MS
Jim R. Lowe, MS
Tom Harris, MS
Brian Nussenbaun, MD

***speaker**

Notes

29. 9:28 a.m. Bioelectronic Microphone for a
Totally Implantable Cochlear Implant
Anthony J. Maniglia, MD*
Taraneh Azar, MD
Hassan Abbass, MD
Wen H. Ko, PhD
Steven L. Garverick, PhD
Philip J. Amantia

9:36-9:51 a.m. **Focused discussion on preceding papers**

9:51 a.m. **Break**

Endolymphatic Hydrops

30. 10:15 a.m. Dysautonomia as an Etiology of
Meniere's Syndrome. A Review of
55 Cases
Dennis G. Pappas, Jr., MD*
Dennis G. Pappas, Sr., MD
Phillip C. Watkins, MD
31. 10:23 a.m. Salt Load Electrocochleography
William L. Meyerhoff, MD, PhD*
Angela G. Shoup, PhD
Bradford A. Gamble, MD
32. 10:31 a.m. Low Dose Methotrexate for the
Treatment of Bilateral Meniere's
Disease
Jefferson K. Kilpatrick, MD*
Aristides Sismanis, MD, FACS
Robert F. Spencer, PhD
Christopher M. Wise, MD
Elias M. Michaelides, MD

***speaker**

Notes

33. 10:39 a.m. The Use of Middle Ear Sustained Release Vehicles to More Appropriately Target Inner Ear Disease
Michael E. Hoffer, MD*
Richard D. Kopke, MD
Derin Wester, PhD
Michael J. O'Leary, MD

34. 10:47 a.m. Selective Labyrinthectomy in Experimental Endolymphatic Hydrops
Paul S. Bennet, MD*
Patrick J. Antonelli, MD
Melanie Adamczyk, MD

10:55 a.m. **Discussion**

Inner Ear Fluids, Ototoxicity

35. 11:03 a.m. Ototoxicity Resulting from Combined Administration of Metronidazole and Gentamicin
Landon C. Riggs, MD*
Anil Shah
William P. Shofner, PhD
M. Rita Young, PhD
Timothy C. Hain, MD
Gregory J. Matz, MD
36. 11:11 a.m. Recovery From Aminoglycoside Ototoxicity???
- F. Owen Black, MD***
Steven W. Wade, MSc
Susan C. Pesznecker, RN

***speaker**

Notes

37. 11:19 a.m. Intracochlear Perfusion with NO-Donators
and NOS-Inhibitors in Guinea Pigs
Katrin Gosepath, MD*
Ulrich Ecke, MD
Wolf J. Maann, MD, PhD, FACS
- 11:27 a.m. **Discussion**
38. 11:35 a.m. The Effects of Stress-Related Hormones
on Inner Ear Homeostasis and Function
Steven K. Juhn, MD*
John Y. Kim, MD
Rick M. Odland, MD
39. 11:43 a.m. Biochemical Markers for Identification
of Human Perilymph
Steven A. Telian, MD*
Michael J. Disher, MD
Quan Sun, PhD
Phillip C. Andrews, PhD
40. 11:51 a.m. Beta-2 Transferrin Assay in the
Identification of Perilymph
Craig A. Buchman, MD*
William M. Luxford, MD
Barry E. Hirsch, MD
Michael J. Fucci, MD
Robert H. Kelly, PhD
- 11:59 a.m. **Discussion**

***speaker**

Notes

12:07 p.m. Closing Remarks
Charles M. Luetje, MD

Introduction of New President
Gregory J. Matz, MD

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- 1991 Gulya, Julianna1558 North Colonial Terrace
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- 1987 Harker, Lee A.....Boystown National Research Hospital
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- 1987 Harner, Stephen G.....Mayo Clinic
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- 1988 Harris, Jeffery P.....9350 Campus Point Drive, 0970
LaJolla, CA 92037-0970
- 1992 Hart, Cecil W. J.....Loyola University Medical Center
2160 South First Avenue (Bldg 105-1870)
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- 1984 Hawke, W. Michael.....1849 Yonge Street, Ste. 10
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- 1996 Hirsch, Barry E.....Eye and Ear Institute Bldg.
200 Lothrop St. - Suite 500
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- 1992 Hoffman, Ronald A.....1430 Second Avenue - Suite 110
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- 1984 House, John W.....2100 West Third
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- 1987 Hughes, Gordon B.....Dept of Otolaryngology
One Clinic Ctr. A-71
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- 1992 Jackler, Robert K.....Univ of California-San Francisco
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- 1990 Jackson, C. Gary.....The Otolaryngology Group
300 20th Avenue, North Suite 502
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- 1992 Jahn, Anthony.....556 Eagle Rock Avenue
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- 1982 Jahrsdoerfer, Robert A..... Dept. of Otolaryngology
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- 1987 Jenkins, Herman A.....Dept of Otolaryngology
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- 1990 Jung, Timothy K.....3975 Jackson St., Suite 202
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- 1988 Kameroner, Donald B.....Eye and Ear Hospital
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- 1991 Kartush, Jack.....Michigan Ear Institute
27555 Middlebelt Road
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- 1992 Katsarkas, Athanasios.....Royal Victoria Hospital - #E4.48
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- 1991 Konrad, Horst.....Southern Illinois University
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- 1993 Kumar, Arvind.....1855 W. Taylor St., M/C 648
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- 1993 Lesinski, S. George.....10550 Montgomery Road
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- 1987 Lindeman, Roger C.....1100 Ninth Avenue - #900
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- 1991 Luetje, Charles M.....Otologic Center, Inc.
Penntower Office Center
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- 1987 Mangham, Jr., Charles A.....Seattle Ear Clinic
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- 1997 McElveen, John T.....3404 WakeForest Road, Ste 303
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- 1981 Meyerhoff, William L.....Univ of Texas Health Science Ctr
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- 1987 Miyamoto, Richard T.....702 Barnhill Drive - Ste. 860
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- 1988 Nadol, Jr., Joseph B.....243 Charles Street
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- 1987 Nedzelski, Julian M.....Sunnybrook Med. Ctr. - Dept. of Otolaryn.
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- 1985 Neely, J. Gail.....Washington University School of Med.
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- 1995 Nelson, Ralph A.....House Ear Institute, Inc.
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- 1995 Niparko, John P.....Dept. of Otolaryngology-HNS
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- 1988 Pillsbury, Harold C.....610 Burnett-Womack Bldg, CB7070
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- 1992 Roland, Peter S.....Department of Otolaryngology
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Dallas, TX 75235-9035
- 1997 Rubin, Allan M.....Medical College of Ohio Hosp
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- 1989 Rybak, Leonard P.SIU School of Medicine
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- 1992 Sasaki, Clarence T.....Yale Univ. School of Medicine
Section of Otolaryngology
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- 1990 Sataloff, Robert T.....1721 Pine Street
Philadelphia, PA 19103
- 1983 Schindler, Robert A.....400 Parnassus Avenue
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- 1995 Schleuning, Alexander3181 S.W. Sam Jackson Park Road
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- 1990 Schuring, Arnold G.....3893 East Market Street
Warren, OH 44484
- 1993 Schwaber, Mitchell.....703 Overton Park
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- 1967 Shea, Jr., John J.....6133 Poplar Pike
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- 1995 Shelton, Clough.....50 North Medical Drive 3C120
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- 1973 Silverstein, Herbert.....1961 Floyd Street, Suite A
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- 1972 Singleton, George T.....University of Florida JHMHC
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- 1988 Smith, Peter G.....Midwest Otologic Group
621 South New Ballas Road
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- 1979 Spector, Gershon Jerry.....517 South Euclid Ave.
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- 1997 Telian, Steven A.....Dept. of Otolaryngology-HNS
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- 1996 Todd, Jr., N. Wendell.....1052 Castle Falls Drive
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- 1997 Wackym, Phillip A.....Dept. of Otolaryngology
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- 1993 Wazen, Jack J.....Columbia University
630 W. 168th St.
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- 1990 Weider, Dudley J.....38 Rip Road
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- 1987 Wiet, Richard J.....950 York Road
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- 1992 Wilson, David F.....911 N.W. 18th Avenue
Portland, OR 97209
- 1996 Yanagisawa, Eiji.....98 York Street
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- 1994 (1969) Bailey, Jr., H.A.Ted.....9601 Lile Drive
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- 1990 (1958) Bellucci, Richard J.....162 East 71st Street
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- 1988 (1961) Bradley, Wesley H.....13 Saybrook East
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- 1988 (1964) Brockman, Seymour J.....222 S. McCarty Dr.
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- 1994 (1969) Buckingham, Richard A.....145 Northwest Highway
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- 1992 (1972) Caparosa, Ralph J.....420 E. North Avenue #402
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- 1996 (1975) Catlin, Francis I.....13307 Queensbury Lane
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- 1994 (1973) Chandler, J. Ryan.....1700 NW 10th Avenue
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- 1992 (1969) Cody, D. Thane.....541 LeMaster Dr.
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- 1990 (1966) Cole, James M.....1301 Red Ln
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- 1989 (1958) Derlacki, Eugene L.....Northwestern Med. Faculty Fdn.
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- 1992 (1972) Hilding, David A.....3156 So. Plateau Dr.
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- 1990 (1970) Hohmann, Albert.....3154 Shoreline Lane
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- 1990 (1960) Hough, Jack V.....3400 NW 56th Street
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- 1975 (1947) House, Howard P.....2100 West Third Street
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- 1995 (1964) House, William F.....Newport Lido Medical Center
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- 1975 (1953) Jordan, Raymond E.....P.O. Box 7
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- 1975 (1997) Montgomery, William.....243 Charles Street
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- 1978 (1957) Myers, David.....2700 N. Forest Rd. #123
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- 1994 (1974) Myers, Eugene.....Eye and Ear Institute
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- 1994 (1988) Nager, George T.....Johns Hopkins Hosp.-Dept. of Otolaryn.
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- 1993 (1968) Naunton, Ralph F.....DCSD-NIDCD EPS-400B
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- 1993 (1973) Pennington, Claude L.....PO Box 1916
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- 1992 (1975) Powers, W. Hugh.....728 Wind Willow Way
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- 1983 (1958) Rambo, J.H. Thomas.....150 East 77th Street
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- 1993 (1972) Ritter, Frank N.....2675 Englave Drive
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- 1991 (1969) Robinson, Mendell.....130 Waterman Street
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- 1972 (1997) Ronis, Max L.....3400 North Broad St.
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- 1996 (1974) Ruben, Robert.....Montefiore Medical Center
111 East 210th St. (VCA-4)
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- 1992 (1967) Rubin, Wallace.....3434 Houma Boulevard
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1993 (1973) Snow, Jr., James B.....	33506 Tuckahoe River Road Easton, MD 21601
1990 (1967) Stroud, Malcolm H.....	(address unknown)
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1990 (1961) Tabb, Harold G.....	1430 Tulane Avenue New Orleans, LA 70112
1985 (1965) Taylor, G. Dekle.....	13500 Mandarin Road Jacksonville, FL 32223
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1986 (1964) Withers, Ben T.....	4703 Ivanhoe Houston, TX 77027
1994 (1971) Wolfson, Robert J.....	1920 Chestnut Street Portland, OR 97201
1987 (1964) Wright, William K.....	3671 Delmonte Houston, TX 77019

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- 1973 Fernandez, Cesar MD.....University of Chicago
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- 1997 Jastreboff, Pawel J., PhD.....Uni of Maryland Schl of Medicine
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- 1962 Smith, Catherine A. PhD.....16200 Pacific HWY. #34
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- 1970 Valvassori, Galdino MD.....697 Sheridan Rd.
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- 1987 Van De Water, Thomas MD.....Albert Einstein College of Med.
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- 1974 Vernon, Jack A. PhD.....3515 S.W. Sam Jackson Park Rd.
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- 1984 Zwislocki, Jozef J. ScD.....Institute of Sensory Research
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- 1996 Thomsen, Jens, M.D.....ENT Department
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DK-2900 Hellerup, DENMARK
- 1995 Booth, Mr. J. Barton.....18 Upper Wimpole Street
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- 1996 Moffat, Mr. David A.....Dept. Of Otoneurological and
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- 1995 Causse, Jean-Bernard, M.D.....Traverse de Beziers
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- 1996 Mann, Wolf J., M.D.....University ENT Department
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- 1995 Bagger-Sjoberg, Dan, M.D.....Dept. Of Otolaryngology
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- 1997 Pyykko, Ilmari, M.D.....Professor-ENT Department
Karolinska Hospital
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- 1996 Rask-Andersen, Helge, M.D., Ph.D.....Stigbergsvagen 11, 752 42
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- 1997 Antarasena, Soontorn, M.D.....Chairman, Dept of Otolaryngology
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- 1993 Albermaz, Pedro.....4405 N.W. 73rd Ave.
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- 1993 Babal, Aziz..... Neurotology Section, ORL Department
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Alexandria, EGYPT
- 1993 Chiossone, Edgar.....25897 E30 Aartado 62-277
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- 1985 Fisch, Ugo.....Forchstrasse 26
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- 1992 Goldstein, Jerome C.....1200 N. Nash St. Apt. 1138
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- 1997 Hitselberger, William E.....222 Oceanview Suite 119
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- 1968 Jongkees, L.B.W.....Reijnier Vinkeleskade 71
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- 1985 Morrison, Andrew....."Dyers", Marden Ash
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- 1992 Nomura, Yasuya.....Dept of Otolaryngology
Showa University 1-5-8
Hatanodai, Shinagawa-ku
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- 1983 Portmann, Michel.....114 Ave de'Ares
33000 Bordeaux, FRANCE 33074

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Gunnar O. Proud, M.D.
Active Membership 1959
Senior Membership 1983
Died: March 19, 1997

Cary N. Moon, Jr., M.D.
Active Membership 1965
Senior Membership 1989
Died: May 19, 1997

Robin P. Michelson, M.D.
Active Membership 1974
Senior Membership 1990
Died: June 27, 1997

Jules Waltner, M.D.
Active Membership 1962
Senior Membership 1981
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Claude C. Cody III, M.D.
Active Membership 1958
Senior Membership 1990
Died: November 14, 1997

F. Blair Simmons, M.D.
Active Membership 1973
Senior Membership 1995
Died: February 13, 1998

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1868-69	E. Williams, MD	1939	Isidore Friesner, MD
1870-73	H.D. Noyes, MD	1940	Horace Newhart, MD
1874-76	D.B.St.John Roosa, MD	1941	George M. Coates, MD
1877-78	C.J. Blake, MD	1942	L. M. Seydell, MD
1879-80	A.H. Buck, MD	1943-44	W.C. Bowers, MD
1881-83	J.O. Green, MD	1945-46	Gordon Berry, MD
1884-85	C.H. Burnett, MD	1947	William E. Grove, MD
1886-89	J.S. Prout, MD	1948	B.J. McMahon, MD
1890	O.D. Pomeroy, MD	1949	Marvin F. Jones, MD
1891-94	Gorham Bacon, MD	1950	Philip E. Meltzer, MD
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1900-02	H.G. Miller, MD	1952	Gordon D. Hoople, MD
1903-05	B. Alex Randall, MD	1953	A.C. Furstenberg, MD
1906-07	Emil Gruening, MD	1954	Frederick T. Hill, MD
1908	C.J. Kipp, MD	1955	D.E.S. Wishart, MD
1909-10	Frederick L. Jack, MD	1956	William J McNally, MD
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HEARING AFTER LASER STAPEDOTOMY WITH PRESERVATION OF THE STAPEDIUS TENDON

Herbert Silverstein, M.D., Seth I. Rosenberg, M.D., T. Oma Hester, M.D.

After stapes surgery, many patients will complain of hyperacusis. Presumably this is due to the stapedius tendon transaction during the procedure. Since there is no treatment and most are happy to obtain a hearing gain, patients learn to live with the symptom of hyperacusis. Using the argon laser with a hand-held probe, the stapedius tendon can be preserved during stapedotomy with or without a prosthesis.

To evaluate the benefits of preserving the stapedius tendon, the following tests were performed and the results evaluated before and after stapedotomies with and without preservation of the stapedius tendon: speech discrimination while masking the ipsilateral ear, establishing the uncomfortable loudness level, and acoustic reflex. Questionnaires were sent to both sets of patients to determine how noise affects the patient's operated ear.

The advantages of preserving the stapedius tendon include: stabilization of the incus during insertion of the prosthesis, protection of the inner ear from barotrauma and acoustic trauma, reduction of hyperacusis, and allowing the patient to hear better in a noisy environment.

It is suggested that preserving the stapedius tendon is beneficial and technically possible in stapes surgery.

ANESTHESIA FOR STAPEDECTOMY

Jack J. Wazen, M.D., Beth Wambach, M.D.

Arlene Markowitz, M.D.

Stapedectomies have been traditionally performed under local anesthesia in order to carefully monitor the patient's hearing or the onset of dizziness suggestive of significant vestibular stimulation.

We have reviewed the results of 154 stapedectomies performed on 140 patients between January 1984 and September 1995. General anesthesia was used in 76 patients and local anesthesia in 78. The two groups of patients were compared as to their hospital length of stay, the incidence of post-operative vertigo or dizziness, nystagmus, nausea and vomiting, as well as their hearing improvement. No significant differences were found between both groups in any of the above parameters.

The type of anesthesia used did not appear to influence the outcome in stapes surgery. General anesthesia did not carry with it any increased risks of otologic complications. Its use may be even more practical in training programs where longer operating times are expected.

EXPERIENCE WITH STAPES SURGERY IN A LARGE TEACHING INSTITUTION: THE ROLE OF THE STAFF SUPERVISING SURGEON IN OUTCOMES

Peter C. Bondy, M.D., Lorenz F. Lassen, M.D., F.A.C.S.

Introduction: This review examines the residency experience with stapes surgery at a large teaching institution. We compare the results of our residents' stapes experience with that of other major academic institutions and examine what factors impact on the success of stapes surgery at our institution.

Methods: This is a retrospective review of 58 otolaryngology department records for stapes surgery done between 1986 and 1996. Air-bone gap and pure tone average were measured using the frequencies of 500, 1000, and 2000 hertz (Hz). Postoperative closure of the air-bone gap to within 10 dB or less is the yardstick for a successful operative procedure, so a two-tailed chi square test was used to compare hearing results among covariants.

Results: 38 (66%) of 58 patients undergoing primary stapes surgery had postoperative air-bone gap closure to within 10 dB. When stapes surgery was supervised by fellowship-trained otologists, 25 (81%) of 31 patients had a successful result. When residents were supervised by general staff otolaryngologists, 13 (48%) of 27 patients had successful outcomes. The difference in successful outcome for primary stapes surgery between residents staffed by fellowship-trained otologists and residents supervised by general otolaryngologists was statistically significant ($p=0.023$).

Conclusions: The best postoperative hearing results are obtained when residents are supervised by fellowship-trained otologists.

STAPEDECTOMY FOR FAR-ADVANCED OTOSCLEROSIS

Paul F. Shea, M.D., Xianxi Ge, M.D., John J. Shea, Jr., M.D.

Purpose: To present the results of stapedectomy for far-advanced otosclerosis and describe the differential diagnosis of far-advanced otosclerosis as distinct from sensorineural hearing loss (SNHL) of other causes.

Methods: Stapedectomy was performed on 85 ears of 65 patients with far-advanced otosclerosis. The pure-tone average (PTA) was calculated from 500 to 2000 Hz. Improved hearing was defined as closure of the air-bone gap to 10 dB or less, and/or an air conduction improvement of at least 20 dB.

Results: The 256 Hz magnesium tuning fork revealed air conduction (AC) < bone conduction (BC) in 79 of 85 ears (92.9%). Overall, hearing improvement was achieved by stapedectomy in 58 of the 85 ears (68.2%). In Group 1, defined as an AC PTA > 90 dB and a BC PTA > 60 dB, hearing improved in 31 of 35 ears (88.6%). In Group 2, defined as AC PTA > 90 dB and nonmeasurable BC, hearing improved in 13 of 18 ears (72.2%). In Group 3, defined as nonmeasurable AC, and BC PTA > 60 dB, improvement was seen in 2 of 5 ears (40%). In Group 4, defined as nonmeasurable AC and BC, improvement was seen in 12 of 27 ears (44.4%).

Conclusions: The 256 Hz magnesium tuning fork is the most important diagnostic tool in differentiating far-advanced otosclerosis from SNHL of other causes. Stapedectomy improves hearing in a high percentage of patients with far-advanced otosclerosis, especially when they have measurable hearing by air conduction.

THE EFFECT OF GELFILM IN THE PREVENTION OF FIBROSIS IN THE ANIMAL MODEL

Michael A. McGhee, M.D., John L. Dornhoffer, M.D.

The use of Gelfoam in the middle ear space has long been performed as a vehicle for hemostasis as well as a support structure to stabilize prostheses and support tympanic membrane grafts. It has, however, been demonstrated in the animal model that using Gelfoam in the setting of disrupted or excoriated mucosa produces fibrosis that potentially could form scar bands that theoretically would effect aeration of the middle ear space and effect the maximum hearing level that could be achieved. Many substances have been used in the middle ear space to protect the mucosa including silastic, teflon and Gelfilm. It has been necessary in this day of cost management to develop materials that are accepted by the body as well as degraded so that removal is unnecessary. Gelfilm has been shown to fulfill this need. No studies to date have combined Gelfoam and Gelfilm in the same animal to demonstrate the barrier effect of Gelfilm to fibroblast migration, to show prevention of fibrosis in the mastoid cavity after otologic surgery when Gelfoam and Gelfilm are used together and to demonstrate that Gelfoam can safely be used in the middle ear when Gelfilm is used to protect the mucosa. Adult male Mongolian Gerbils were divided into two groups of twelve. After excoriating the mucosa of the superior mastoid bulla with an otologic instrument, either Gelfoam or Gelfoam/Gelfilm was applied to the cavity. The animals are currently being evaluated at 2, 4 and 8 weeks postoperatively. Preliminary data currently shows a significant decrease in the gross appearance of fibrosis in the mastoid bulla when Gelfilm is used to protect the mucosa. Histologic studies are currently underway to evaluate the objectives of this study.

EPITYMPANIC APPROACH TOWARDS CHOLESTEATOMA

John L. Dornhoffer, M.D., F.A.C.S.

Introduction: The main issue regarding cholesteatoma surgery centers around handling the posterior canal wall. The canal wall up (CWU) procedure preserves the normal anatomy, thus avoiding bowl problems and the necessity for periodic cleaning. However, this technique is associated with a 20-40% recurrence rate. Canal wall down (CWD) techniques offer a lower recurrence rate (5%-10%) but at the expense of creating a mastoid bowl, which requires periodic cleaning and may result in medical dependency. The novel epitympanic approach offers the advantages of both CWU and CWD surgeries. In this single-stage procedure, the epitympanum is widely exposed and the canal wall is partially removed, allowing complete exposure of the cholesteatoma. The canal wall is then reconstructed with cartilage from the Cymba area of the conchal bowl.

Method: A computerized otologic database was used to identify those patients who had undergone cholesteatoma removal with the epitympanic approach with at least 1-year follow-up. A retrospective chart review was performed to assess outcome with regard to hearing and recurrence.

Results: Between 1994 and present, the epitympanic approach was performed on 97 patients, 76 of whom had 1-year follow-up. Recurrent cholesteatoma was found in 2 patients and a deep retraction in a third patient. The average post-operative pure tone average air-bone gap was significantly improved (12.6 dB versus 26.4 dB pre-operative, $p < 0.05$).

Conclusion: The epitympanic approach offers the wide exposure of CWD surgery while preserving the normal anatomy as in CWU surgery. Preliminary results show excellent hearing with a low recurrence rate, offering the possibility of single-stage surgery.

SUPRALABYRINTHINE APPROACH TO THE PETROUS APEX

Fred F. Telischi, M.D., Michal Luntz, M.D., Michelle L. Whiteman, M.D.

Of the various approaches to the petrous apex, the supralabyrinthine dissection has been the least described. A case of an eleven month old infant with petrous apex abscess drained through the supralabyrinthine air cells prompted an anatomic study of the dimensions of this approach. Fifteen temporal bones were dissected to completely expose the epitympanum. This required mastoidectomy, exenteration of zygomatic root and epitympanic air cells, and removal of the incus. Measurements were taken from three sides of a triangle described by the tympanic facial nerve (TFN), superior semicircular canal (SSCC) and tegmen tympani (TT). Mean lengths were 5.5 millimeters (TFN), 5.2 mm (SSCC) and 7.7 mm (TT). Petrous apex air cells were accessible from the supralabyrinthine approach in all specimens. Also, computerized tomographic scans of normal human temporal bones were studied to evaluate the imaging characteristics of this anatomic area. We conclude that the supralabyrinthine approach provides adequate access to the petrous apex for drainage and biopsy in selected cases.

LONG-TERM MIDDLE EAR VENTILATION DURING TYMpanoplasty WITH A SUBANNULAR T-TUBE

Timothy O'Hare, M.D., Ph.D., Joel A. Goebel, M.D., F.A.C.S.

Introduction: Chronic hypoventilation of the middle ear leads to a myriad of complications, such as adhesive otitis media, atrophic tympanic membranes, and failure when performing tympanoplasty. The main treatment for chronic hypoventilation is long-term ventilation. Therefore, we have developed a simple technique for insertion of a subannular T-tube anteriorly. A prior pilot study demonstrated the advantages of this technique. We now report on a larger series of patients with longer follow-up.

Methods: A series of 16 consecutive patients with the diagnosis of eustachian tube dysfunction (ETD) and/or adhesive otitis media and who underwent tympanoplasty were included in the study. Simultaneously a T-tube was placed anteriorly in a subannular position. At routine follow-up we have assessed the position and patency of the T-tube, tympanic membrane retraction, hearing result, and complications.

Results: There were 16 patients and ears that received a subannular tube. Thirteen ears had ETD, and 7 had adhesive otitis media. All patients had prior surgery. All patients underwent tympanoplasty, 3 also had a simple mastoidectomy, and 10 patients underwent ossiculoplasty. The mean follow-up is 7 months. All 16 patients have a patent tube that has not migrated or been extruded. 70% of patients tested had improved hearing. Complications were minimal.

Conclusions: We describe a technique for long term ventilation of the middle ear at the time of tympanoplasty. It offers the advantages of ease of placement, use when there is tympanic membrane loss, and use during simultaneous tympanoplasty.

ABR HEARING SCREENING FOR HIGH-RISK INFANTS

Paul R. Kileny, Ph.D., Lori A. Van Riper, M.S.

Since 1987, newborns presenting with one or more risk factors for hearing loss have undergone ABR hearing screening at our institution. Of the total of 1971 infants evaluated, 374 (19%) failed the initial screening. Of those, 130 infants (6.6%) were diagnosed with significant hearing loss. One-hundred eight infants presented with bilateral hearing loss and 22 with unilateral hearing loss. The severity of hearing loss ranged from mild (54 infants or 42%) to moderate and moderately severe hearing loss (40 representing 42%) to severe and profound hearing loss (36 infants representing 27%). In ten of the 76 children with greater than moderate hearing loss, there was a delayed onset of hearing loss. There was a trend for children with a greater hearing loss to present with more risk factors (three or more). The two risk factors that were the most predictive of hearing loss were respiratory problems at or shortly after birth and craniofacial anomalies. Of the 108 infants with bilateral hearing loss 62 (47%) experienced respiratory problems. Of the 140 infants whose risk factors included craniofacial anomalies 44 (31%) were diagnosed with hearing loss. Our results strongly support the importance of early, prospective hearing screening of infants with known risk factors for hearing loss. A rigorous system for follow up is also critical especially given the likelihood of progressive or late onset hearing loss in this population.

SENSORINEURAL HEARING LOSS FOLLOWING OCCLUSION OF THE ENLARGED VESTIBULAR AQUEDUCT

D. Bradley Welling, M.D., Patrick W. Slater II, M.D., Michael D. Martyn, M.D.

Bruce J. Gantz, M.D., William M. Luzford, M.D., Clough Shelton, M.D.

Surgical occlusion of the enlarged vestibular aqueduct has recently been reported in an attempt to stabilize the fluctuant and progressive sensorineural hearing loss associated with this condition. Wilson et al.¹ first reported an intraluminal sac occlusion technique with stabilization of hearing reported in 6 of seven patients. Welling et al.² showed continued hearing loss in 10 patients with extraluminal compression of the enlarged sacs. Although the average rate of hearing loss was retarded, there was no overall statistically detectable stabilization in the operated ears when compared to controls.

This report adds an additional 8 surgical patients to the literature, 6 with intraluminal and 2 with extraluminal duct occlusion. Unfortunately, 5 of 8 patients (62.5%) sustained significant sensorineural hearing loss. Differences in patient selection, surgical technique and the natural course of the disease are discussed. The otologic surgeon is alerted to potential sensorineural hearing loss following occlusion of the enlarged vestibular aqueduct.

1. Wilson DF et al. Endolymphatic sac obliteration for large vestibular aqueduct syndrome. *Am J of Otol*, 18: 101-106, 1997.
2. Welling DB et al. Endolymphatic sac occlusion for the enlarged vestibular aqueduct syndrome. *Abstract Am Neurotol Soc*.

INFLUENCE OF MITOCHONDRIAL METABOLITE SUPPLEMENTS ON AGE-RELATED HEARING LOSS

Michael D. Seidman M.D., Mumtaz J. Khan, M.D.

Uma Bai, Ph.D., Najeeb Sherwany, M.D.

The purpose of this study was to assess the effects of mitochondrial metabolites, (α -lipoic acid and acetyl L-carnitine, on aging and on the preservation of age-related hearing loss. These metabolites upregulate and enhance mitochondrial function. Reactive oxygen metabolites (ROM) such as singlet oxygen and hydroxyl radicals, are known products of oxidative metabolism and are constantly being generated endogenously (Wallace, 1992). The Membrane Hypothesis of Aging (NM) suggests that aging is the result of progressive insults by these reactive oxygen metabolites on mitochondria and other cellular structures. With time, these oxidative insults accumulate contributing to cellular demise and resultant senescence.

These experiments investigate the effects of (α -lipoic acid and acetyl L-carnitine in aged (>24 months) Fisher rats by measuring hearing sensitivities with Audiometric Brainstem Responses (ABR), and examining specific age-associated mitochondrial DNA (mtDNA) deletions with Polymerase Chain Reaction (PCR). A statistically significant effect was seen with acetyl L-carnitine in the preservation of age-associated hearing loss. Additionally, (α -lipoic acid also showed an obvious trend in maintaining hearing thresholds. Preliminary PCR data showed decreased amplification of the common aging deletion (4834bp) in subjects treated with acetyl L-carnitine, when compared to the control. These results suggest that mitochondrial metabolites may influence age associated hearing impairment by up-regulating mitochondrial function.

PROGRESSIVE SENSORINEURAL HEARING LOSS, SUBJECTIVE TINNITUS AND VERTIGO CAUSED BY DIABETES MELLITUS

Jack L. Pulec, M.D., Marlene B. Pulec, Ignacio Mendoza H., M.D.

The otologist frequently sees patients with progressive sensori-neural hearing loss, subjective aural tinnitus and vertigo with no apparent cause. Uncontrolled diabetes mellitus with elevated blood sugar may be the cause of inner ear malfunction on a biochemical basis. Four thousand new patients, 2,000 of whom had complaints of sensori-neural hearing loss, subjective tinnitus or vertigo, were studied. All had a complete neuro-otologic examination and appropriate audiometric and vestibular tests and imaging. In addition, blood tests were performed which included 5hr. glucose tolerance tests and lipid phenotype studies. Almost 5 per cent were found to have abnormally high blood sugar associated with a diagnosis of diabetes mellitus. The otologic characteristics will be described. Strict dietary control and medical treatment can lead to a significant improvement in many cases after seven days. The results of the treatment will be given.

HISTOLOGIC CHANGES OF THE COCHLEA AFTER AUTOMOBILE AIR BAG DEPLOYMENT

Douglas E. Mattox, M.D., Weihua Lou, M.D.

Joel Kalb, Ph.D., C. Richard Price, Ph.D.

Introduction: Reports of hearing loss after automobile air bag deployment have been infrequent despite the high sound pressure levels generated by air bags; estimated in excess of 170 dB. This resilience of the ear to high intensity sound exposure is predicted by the Price Kalb model of the ear, based primarily on the sound pressure wave reaching the elastic limit of the annular ligament .

Methods: Anesthetized cats were placed in a truck cabin and exposed to deployment of driver and/or passenger air bags. The rising time was 20 ms, the duration of the propellant combustion was 80 ms and total inflation time was 100 ms. Noise impulse waveforms were recorded with a microphone. ABR's at 1, 2, 4, 8, and 16kHz were performed before, after, and one and six months after exposure. At six months surface preparations and cytochleograms prepared of the ears.

Results: Compound threshold shift immediately and one month after the exposure averaged 60 dB at 4kHz, and a permanent threshold shift of 35 to 40 dB remained at six months. Most animals showed a threshold shift at all frequencies, greatest at 4kHz. The histologic damage centered at 12 mm from the base (total length averaged 25mm). The inboard ear, closest to the sound source, consistently suffered more hearing loss and histologic damage than the out board ear, which was protected by head shadow effects.

Conclusions: Based on these experiments, it would appear that anesthetized cats are more susceptible to cochlear injury from air bag deployment than the "average" human.

DOES OTOSCLEROSIS OCCUR ONLY IN THE TEMPORAL BONE?

Pa-Chun Wang, M.D., Saumil N. Merchant, M.D., Michael J. McKenna, M.D.

Robert S. Glynn, Sc.D., Joseph B. Nadol, Jr., M.D.

It is widely assumed that otosclerosis is confined to the temporal bone (TB) and does not occur elsewhere in the skeleton. However, this assumption has never been tested. It is important to investigate this issue, particularly in light of evidence that otosclerosis may be a systemic disease which might be expected to affect other bones. There is also recent evidence suggesting a common genetic etiology between mild forms of osteogenesis imperfecta and otosclerosis with mutations in the COL IA 1 gene.

We conducted a light microscopic study on 2 patients with otosclerosis involving the TB to determine whether other skeletal bones show histologic evidence of otosclerosis. In each patient, 10 skeletal endochondral bones were sampled (base of skull, skull cap, rib, vertebra, scapula, humerus, radius, femur, pelvis and tibia). Seventy-seven % of 171 TB sections showed otosclerosis. The mean surface area of the otic capsule in each TB section was 131 MM². None (0%) of 213 non-TB skeletal sections showed otosclerosis. The mean surface area of each skeletal section was 278 mm². The difference between the two groups was significant at $p < 0.001$. Furthermore, our data indicate that with 95% confidence, the true rate of otosclerosis in the skeletal bones of the two individuals examined is 0-3%.

Our findings suggest that otosclerosis does not occur outside the TBs. Thus, in cases of otosclerosis with the same genetic etiology as osteogenesis imperfecta, there must be factors unique to the TB that lead to otosclerosis. These possible factors will be discussed.

THE OTOLOGICAL ASPECTS OF USHER'S SYNDROME: CLASSIFICATION, HISTOPATHOLOGY AND MANAGEMENT

Arvind Kumar, M.D., F.R.C.S. (Edin), Isamu Sando, M.D.

Haruo Takahashi, M.D., Gerald Fishman, M.D., Reena Dhandra, B.A.

Based on our study of the ophthalmologic, audiologic and vestibular findings in patients with retinitis pigmentosa (RP), we reported several years ago, a classification of this disease as Usher's type I and type II. Molecular studies have subsequently delineated the syndrome into corresponding subtypes based on chromosomal location of genetic defects. The temporal bone histopathology of a patient previously studied by us in detail and classified as Usher's type II is presented as an illustration of these clinical findings at the microscopic level. In addition to degeneration of Corti's organ, both ears showed a moderate reduction in the spiral ganglion population by 30-50%. Previous histopathologic reports of 3 RP patients showed similar abnormalities. However, earlier reports in the German literature on four pediatric patients, who presumably were of the type I variety, showed severe reduction of the spiral ganglion population. Should such children receive a cochlear implant? On an empirical basis we implanted three children, two of whom are "star performers." The third child has made progress but is slower. Obviously the implant works even in the face of a presumed reduced cochlear neuronal population. We feel that in the third child, family and environmental circumstances are the reasons for his suboptimal performance. Based on these results, we conclude that other things being equal, deaf RP children should be offered the cochlear implant.

Objectives

1. To review from a historical context the classification of Usher's syndrome and compare it to the classification scheme that is used commonly today to make a clinical diagnosis of Usher's syndrome.
2. To identify the scientific techniques behind studies that define the molecular defect in Usher's syndrome and to discuss their current use in defining subtypes of the disorder as well as their potential utility in diagnosis.
3. To report the temporal bone histopathology of an Usher's type II patient and to report the results in three Usher's patients who were implanted at our center in the last five years

**CONSTRUCTION AND CHARACTERIZATION OF A HUMAN
ACOUSTIC NEUROMA (VESTIBULAR SCHWANNOMA) cDNA
LIBRARY***

Phillip A. Wackym, M.D., Elizabeth Toh, M.D., Marta Troyanovskaya, Ph.D.

We constructed a complementary DNA (cDNA) library from human acoustic neuroma (vestibular schwannoma) tissue as part of our ongoing studies of the molecular biology of auditory and vestibular function and dysfunction. Such tissue-specific recombinant cDNA libraries provide a reverse molecular genetic approach to identify and clone unknown genes.

For construction of the cDNA library, we obtained approximately 3 ml of fresh tumor tissue removed during the resection of a 4 cm acoustic neuroma in a patient with neurofibromatosis type 2 (NF2). Poly(A)⁺ RNA was isolated from total cellular RNA. Oligo(dT) primers were used to synthesize the cDNAs using reverse transcriptase and these were unidirectionally inserted in Uni-Zap[®] XR (Stratagene, La Jolla) bacteriophage vectors. 2.4 million primary plaques were obtained. Inserts averaged 1.8 kilobases (kb) in length, and the range was 0.8-3.0 kb.

Screening such a library by any of several different strategies can identify gene sequences expressed in the tissue from which the library was constructed. Most importantly, the library can be used to identify proteins important for inner ear function and dysfunction. Our initial screening effort focused on sequencing 40 randomly selected clones. The sequences obtained were compared to all sequences in the GenBank database. These data have implications for understanding the molecular mechanisms of acoustic neuroma (vestibular schwannoma) tumor biology. In addition, this line of research may lead to novel applications of gene therapy in the management of patients with hearing and balance dysfunction.

*(Supported by NIH/NIDCD DC02971-03)

ELECTRON MICROSCOPIC STUDY ON CYSTIC VESTIBULAR SCHWANNOMA

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Introduction: The cystic variant of Vestibular Schwannoma (VS) is sporadically reported and it is suggested that approximately 2 % of VS develop rapid cystic expansion (Lanser et al, 1992). Cystic elements in VS have been reported to be the result of degenerative changes in type A tissue, specially in large "ancient" tumors. In a previous histopathological study (Charabi et al 1994) we have demonstrated the presence of cyst membranes or membrane-like structures in cystic VS as well as a relatively rapid growth of implanted cystic VS on nude mice (Charabi et al 1994). In the current study we performed an electron microscopic study on solid/cystic human VS and the corresponding implants growing on the athymic nude mice.

Material: VS from 6 patients (4 solid tumors and 2 cystic) were obtained and the tumor specimens were formalin fixed and paraffin embedded for routine histological examinations. Specimens from the 6 human tumors were specially fixed in order to perform the electron microscopic study. Tumor tissue was implanted on 25 athymic nude mice.

Results: The take ratio was 24/25. One specimen vanished and 24 specimens survived. The specimens from the human cystic VS grew on the athymic nude mice producing cystic elements. The ultrastructure of the cystic VS, studied by the electron microscope revealed a significantly different cellular structure compared to the solid tumors.

Conclusion: The results of the current study add another piece of knowledge concerning the cystic variant of VS and support the theory that the cystic elements in VS are not the result of degenerative changes in large "ancient tumors", but due to the ability of distinctive types of Schwann cells which are able to produce cystic elements as observed in our in vivo growth model.

Objectives:

1. To describe the macroscopic and microscopic appearance of the cystic vestibular schwannoma (VS)
2. To describe the ultrastructural appearance of cystic VS
3. To discuss the clinical implication of the special cellular structure of cystic VS

PAROXYSMAL POSITIONAL VERTIGO SYNDROME

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Introduction. This study was initiated in order to investigate the differential diagnosis of patients with benign paroxysmal positional vertigo (BPPV) of different canals' origin.

Methods. The eye movements of eighteen patients were evaluated with the use of infra-red video cameras following Hallpike tests.

Results. The responses of the eighteen patients were typical of the BPPV posterior semicircular canal origin producing disconjugated, torsional, and vertical nystagmus. Three patients converted immediately or shortly after the Epley maneuver to responses attributable to the anterior semicircular canal stimulation. Of these, two responded to stimulation of the plane of the anterior semicircular canal and one to stimulation of both the anterior and posterior semicircular canals. Another three patients converted to responses attributable to horizontal semicircular canal stimulation, producing pure horizontal nystagmus. Two of these were of the canalithiasis variety, i.e., responding to the Hallpike test with geotropic nystagmus, and one was of the cupulolithiasis variety, responding to position (gravitational) changes with antigeotropic nystagmus. One other patient's nystagmus was induced by stimulation of the two vertical canals and of the horizontal semicircular canal (cupulolithiasis).

Conclusion. The location of the BPPV pathology can be diagnosed on the basis of nystagmus reactions to Hallpike tests directed to specific semicircular canals.

PAROXYSMAL POSITIONAL VERTIGO: IDIOPATHIC VS POST-TRAUMATIC

Athanasios Katsarkas, M.D., M.Sc.

In our Dizziness Clinic (N=15,233), 2,525 (15.55%) patients were found to suffer from paroxysmal positional vertigo (PPV). All patients were examined and investigated by the author.

There was no apparent cause in 1,829 patients (idiopathic group). In 1492/1829 patients, the nystagmus was clearly identified. It was compatible with excitation of the posterior semicircular canal in 1,475/1,492 patients (age:56±13, 458 males/1017 females), unilateral in 1,383 cases, bilateral in 92, and compatible with excitation of the horizontal semicircular canal in 17/1,492 cases. In 342 patients (age:49±16, 166 males/176 females) the PPV was post-traumatic; positional nystagmus was clearly identified in 154/342 patients, and was unilateral in 132/154 and bilateral in 22/154 patients.

No case could be attributed to brain stem dysfunction. Statistical comparison of the idiopathic vs the post-traumatic group showed: 1) Patients being older ($p<.001$), with higher prevalence among women than men in the idiopathic group ($p<.001$). 2) Higher prevalence of bilateral occurrence in the post-traumatic group ($p=.005$).

It is concluded that: 1) PPV showed a high prevalence in our Dizziness Clinic. 2) PPV was clearly a peripheral disease. 3) The diagnosis was established by the clinical examination. 4) PPV, attributed to the posterior semicircular canal, was by far the most prevalent. 5) The post-traumatic was different from the idiopathic group in: a) age distribution; b) prevalence among women vs men; c) unilateral vs bilateral occurrence.

**MASTOIDOTOMY TYMPANOTOMY APPROACH FOR
COCHLEAR IMPLANTATION. ADVANTAGES OF THIS
TECHNIQUE. A MULTICENTER MULTINATIONAL STUDY.**

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The surgical aim in multichannel intracochlear implantation is to place the full array of electrodes in the cochlea in a safe and efficient manner. This aim can be achieved using different surgical methods. This paper presents the experience with the mastoidotomy (antrotomy) tympanotomy approach at three implant centers in three different countries. The authors describe their technique, experience, and emphasize the main advantages of this method. To date 78 implants (different types) have been placed with this technique. This approach is technically simple, involves less bone drilling, has no risk to the facial nerve, the active electrode is covered by a thick layer of tissue in all its course, provides a better angle of insertion in the basal turn of the cochlea, and has a faster recovery. It has a small postauricular incision which requires no drains, has less risk of hematoma, and makes healing easier. In addition, it allows direct view and work in the round window niche, as well as sculpturing in cases of ossified cochleas (one case is described in detail). The authors are well aware that to achieve a safe and efficient surgical result, different surgeons might elect different but equally valid approaches. A method is presented, which has worked well for the authors and could be useful to others.

OBJECTIVES.

1. To describe the mastoidotomy (antrotomy) tympanotomy approach for cochlear implantation, and point its main advantages; with special emphasis in sculpturing of the cochlea in case of ossification.
2. To describe the surgical experience with this method in 78 cases performed at three implant centers in three different countries.
3. To describe a surgical method which has worked well for the authors and could be useful to others.

DEEP INSERTION OF COCHLEAR IMPLANT ELECTRODES

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Deep insertion (>20mm) of cochlear implant electrodes has been proposed as a method to improve hearing outcomes by accessing lower frequency areas of the spiral ganglion. One electrode array, which is specifically designed for insertion 30mm or more into the cochlea, is now in clinical trials in the U.S. The potential benefit of deep insertion, however, has not yet been demonstrated.

In order to determine the hearing effects of insertion depth of the Nucleus Mini 22 device, 40 implanted, post-linguistically deafened English speaking adults were retrospectively analyzed. Insertion depths ranged from 17mm (all electrodes inserted) to 25mm (all electrodes plus 10 stiffening rings inserted). Several measures of hearing were analyzed with respect to insertion depth. Statistical analysis demonstrated no significant relationship between insertion depth and hearing results with this device.

MULTICHANNEL COCHLEAR IMPLANTATION IN CHILDREN WITH COCHLEAR OSSIFICATION

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Meningitis is a common cause of profound deafness in children and a large percentage of these children develop ossification of the cochlea. The purpose of this study is to examine the success of cochlear implantation in children with ossification of the cochlea from meningitis.

Between June, 1990 and July, 1997, 88 children with bilateral profound hearing loss not helped by hearing aids have received cochlear implants (Nucleus 22) at our facility. Meningitis was the cause of deafness for 27 (31%). Twenty two (80%) of the children deafened by meningitis had cochlear ossification that was identified by CT scan and confirmed at surgery. One additional child had ossification of unknown etiology.

For 17 (62%) of the children, a partial drill-out of the basal turn of the cochlea was performed followed by complete insertion of all electrodes. Six patients (22%) had extensive ossification requiring circumodiolar drill-out (as described by Gantz) with an average insertion of 18 electrodes. There were no minimal insertions.

In our experience, partial or complete drill-out of the cochlea allows for the complete or near complete insertion of all electrodes with performance that equals or approaches that of patients without ossification. Ossification does not appear to preclude cochlear implantation.

MANAGEMENT OF COCHLEAR IMPLANT INFECTIONS*

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Wendy S. Parkinson, M.A.

Cochlear implant infections are a rare but potentially devastating complication. Removal of an infected device can lead to cochlear fibrosis and ossification potentially requiring a "drill-out" at a later replacement procedure if the contralateral ear is not available for implantation. The expense of these devices can also make replacement difficult if previously available financial resources suddenly become unavailable even temporarily. Excellent speech reception can be suddenly and totally destroyed by a delayed device infection.

We describe our experience with four patients suffering a delayed implant infection. Three of these patients had Ineraid devices where a pedestal complication led to infection. One patient had migration and extrusion resulting in infection of a Cochlear Corporation CI-22 device. The Ineraid patients were managed by revising the pedestal location without manipulating the intracochlear electrode array. The CI-22 patient had his implant removed, rinsed in antibiotic solution and replaced. All patients received a course of intravenous antibiotics.

None of the four patients developed further complications. All maintained or ultimately regained their pre-infection speech reception abilities. Implant infection does not necessarily mandate removal of the device.

*Supported by NIH Grant DC00242

THE NUCLEUS 24 SYSTEM IN CHILDREN

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The Nucleus 24 cochlear implant incorporates an internal system capable of advanced processing, higher rates of stimulation, multiple stimulation modes (monopolar, bipolar, common ground), a removable magnet for MRI compatibility and advanced telemetry capable of recording the VIIIth nerve compound action potential from electrodes within the cochlea. The system also includes a behind-the-ear speech processor.

In compliance with the FDA protocol, subjects were at least 18 months of age, had bilateral profound sensorineural hearing loss and showed a lack of progress with conventional amplification and/or limited speech understanding. Twenty prelingually deafened children ages 20 months-15 years were implanted at our center with the Nucleus 24 system as subjects for this study. Age appropriate pre-implant testing was performed to assess auditory and linguistic skills to determine eligibility. High resolution CT and MRI, where applicable, were done to evaluate temporal bone anatomy. The surgery was performed using the conventional transmastoid, facial recess approach.

There were no major and few minor surgical complications. Upon stimulation, all children received auditory percepts using the SPEAK strategy in a monopolar configuration. Six month postoperative data reveal an improvement from preoperative conditions in auditory perceptual skills and a range of open set speech recognition scores. Data obtained on these subjects will be compared to those reported on other children participating in the clinical trial.

Preliminary results suggest that the Nucleus 24 system offers several advantages over the Nucleus Mini-22 system, is safe and at least as efficacious. Long term results are needed to determine functional superiority.

COMPARISON OF AUDIOLOGIC PERFORMANCE FOLLOWING REIMPLANTATION: A MULTICENTER OVERVIEW

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Dawna Mills, M.A., CCC-A.

This study compares the auditory performance characteristics of adult Nucleus 22 cochlear implant (CI) patients from various CI centers who have been reimplanted with a second Nucleus 22 internal device due to failure of their once functioning original internal device. Variables are compared between the improved, same, and poorer performance groups in order to determine what factors, if any, influence how well patients will do with their replacement CIs.

Through a multicenter chart review and subject questionnaires, the auditory performance and subjective preference between original and replacement CIs in 28 adult subjects was determined. All subjects included in this study had a once functioning original CI prior to failure; with the mean length of original CI use of 28 months. Audiometrically 26% performed better, 47% performed the same, and 26% performed poorer on speech recognition tests. Subjectively 45% of subjects with a once functioning original CI reported that their replacement CI was poorer. Poorer performance could not be correlated with cause of device failure, surgical complications, insertion depth differences, or duration of implant use.

CI internal device failure rates are small (<1%); but with the continually increasing population of implant recipients and the likelihood of future device upgrades, the incidence of CI explantation with subsequent reimplantation will continue. Improved or even consistent performance cannot be guaranteed to patients undergoing replacement surgery due to device failure or upgrade. This study provides important information for counseling experienced CI users that face revision surgery.

COCHLEAR IMPLANT MRI COMPATIBILITY

Noel L. Cohen, M.D., J. Thomas Roland, Jr., M.D.

INTRODUCTION: In all probability, every child in the United States will ultimately require an MRI, illustrating the need for an MRI-compatible device.

Current implants are not MRI-compatible since they contain a magnet. Exposure to MRI would cause pain and possibly device movement. Current might be induced in the implant, causing damage to the cochlea, or the device might be damaged by the current. Finally, a metallic foreign body will produce a signal void on the MRI.

PROCEDURE: Modern implants can be modified by the manufacturer removing magnets and ferro-magnetic parts. This is done for patients expected to require MRI following implantation. The larger problem arises when a recipient needs an MRI some time after implantation. The Nucleus Mini-22 magnet can be removed by a minor surgical procedure, while the magnet in the ABI and the newer CI 24 device is designed to be removable at the time of original surgery or at some later time. Magnets incorporated into devices with ceramic cases cannot be removed at the time of original surgery or subsequently.

In an emergency, MRI can be performed by removing the magnet, removing the implant, or firmly binding the head over the implant, with the hope that no damage will be sustained to the patient or device.

SUMMARY and CONCLUSIONS: All current cochlear implants can be manufactured MRI-compatible, but only the Nucleus 22, 24 and ABI devices can be made MRI-compatible after insertion.

**POSITRON EMISSION TOMOGRAPHY (PET) IN
COCHLEAR IMPLANT RECIPIENTS***

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A number of cortical regions, in addition to the primary auditory cortex, are activated by speech stimuli in normal hearing subjects. The purpose of this study was to determine whether similar networks are activated in profoundly deaf patients who have received a multichannel cochlear implant (CI). PET studies were conducted in 5 normal hearing subjects and 5 CI subjects to measure changes in regional cerebral blood flow evoked by acoustic stimulation. Subjects were blindfolded to eliminate visual input while speech or non-speech stimuli were presented. Scans were obtained for the following conditions: 1) broadband noise; 2) multi-talker babble; 3) multi-syllabic lexical neighborhood test words; and 4) common phrases. Images for the different conditions were obtained and the regions of significant activation mapped in stereotaxic coordinates.

Stimuli perceived as speech by both normal and CI subjects evoked strong bilateral activation in the superior/middle temporal gyri, which are regions that include the primary and secondary auditory cortices. Broadband noise did not significantly activate the auditory cortex. PET promises to provide new insights into the underlying mechanisms of audition through a cochlear implant.

*Supported by NIH-NIDCD

**VARIATIONS IN CENTRAL NERVOUS SYSTEM ACTIVATION
BETWEEN COCHLEAR IMPLANT USERS RECEIVING
MAXIMAL OR MINIMAL BENEFIT**

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This study addresses the underlying variation in performance among cochlear implant users by combining traditional measures of speech perception and speech reading with state-of-the art functional brain imaging with Single Photon Emission Computerized Tomography (SPECT). Considerable variation across cochlear implant users is often reported for open-set speech perception: some individuals receive considerable benefit and others receive minimal benefit. In order to examine possible contributions of the central auditory system to these performances variations, we examined regional cerebral blood flow (rCBF) in individuals with normal hearing, successful cochlear implant users, and cochlear implant users who receive minimal benefit from their implants.

Individuals scoring 80% or greater on open-set speech perception were considered successful users, and individuals scoring 30% or less were considered users with minimal benefit. Subjects were matched by age and gender to normal hearing subject. RCBF was examined during three conditions: visual only (V) presentations, visual plus full auditory map presentations (FA), and visual plus partial, degraded auditory map presentations (DA) in which only channel 13 was stimulated. The V condition serves as the control for the other two states: the difference between V and DA (expressed as [DA-V]) identified auditory activations related to a sound only condition. The difference between the V state and FA state [FA-V] identified all brain areas activated by auditory stimulation for comprehension of the message. Thus, the component of auditory stimulation leading to comprehension was provided by [FA-V] - [DA-V].

Data indicated all primary and associative auditory cortices (Brodmann areas 41, 42, 18, 19, and 22) were activated in normal hearing (NH) subjects during the FA condition. RCBF was blunted in magnitude in NH individuals during the DA condition for areas 41 and 42 with no responses evident in areas 18, 19, and 22. Successful cochlear implant users demonstrated rCBF responses similar to NH individuals for area 41 in the left hemisphere during the FA condition with significant reductions bilaterally during DA conditions. Blunted responses were also observed for areas 42 and 22 in the successful user in both FA and DA conditions. In contrast, the cochlear implant users receiving minimal benefit demonstrated sharply reduced responses in areas 41, 22, and 21.

BIOELECTRONIC MICROPHONE FOR A TOTALLY IMPLANTABLE COCHLEAR IMPLANT

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The purpose of the study is the development of a middle ear bioelectronic microphone (BEM) using the tympanic membrane as a diaphragm and coupled to the currently available implant technology to create a totally implantable cochlear hearing device. Our transducer, which is used for development of a semi-implantable middle ear electromagnetic hearing device (SIMEHD), has received FDA Investigational Device Exemption (IDE) for clinical trials. When this transducer is used in the reverse mode, it functions as a BEM. Acoustic energy applied to the external auditory canal vibrates a neodymium-iron-boron titanium-encased magnet cemented to the ossicles attached to the tympanic membrane. Mechanical energy is transformed into an electric signal by an electromagnetic transducer coil which is positioned in the attic, 0.5 to 1.0 mm away from the magnet. The signal is amplified and transmitted to a speaker as well as to an oscilloscope. A bench model was developed and fresh human temporal bones were tested. Two types of electromagnetic coils were studied: 1) air core; 2) ferrite core. Experiments were carried out using the intact ossicular chain with the magnet cemented to the incus, and with the incus removed and the magnet cemented to the head of the malleus. Both coils showed a flat frequency response. The ferrite coil with a magnet on the malleus had a better millivolt peak-to-peak response by a factor of two as compared with the air core coil in all frequencies. The system is powered by an available transcutaneously R.F. rechargeable implantable disc-shaped lithium 3-volt battery. Chip miniaturization technology of the cochlear implant speech signal processor coupled to a multichannel electrode and to the BEM may lead to a new device applicable to patients suffering from profound and total hearing loss.

DYSAUTONOMIA AS AN ETIOLOGY OF MENIERE'S SYNDROME

A REVIEW OF 55 CASES

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Faulty regulation of the autonomic nervous system or dysautonomia can produce a combination of hyperadrenergic and hypervagal states. This condition can then affect any system of the body, resulting in a multitude of diverse symptoms which include vertigo. There is a subgroup of patients with Meniere's disease that also manifest symptoms of dysautonomia. When conventional treatment for endolymphatic hydrops is initiated, these patients report worsening of their condition. This reflects the hypovolemic state found in these individuals. The records of 55 dysautonomia related Meniere's cases were reviewed. The most frequent non-otologic symptoms reported were fatigue, palpitations, sleep disorders and headache. Physical examination often demonstrated a systolic click on auscultation. Postural blood pressures and heart rates were routinely measured. Electrocochleography was typically suggestive of endolymphatic hydrops bilaterally. The more symptomatic cases were further evaluated by echocardiogram and exercise testing. Treatment focuses on fluid loading and exercise. Patients who demonstrated more hyperadrenergic symptoms were managed with beta-blockers, while those experiencing rather marked hypotension benefited from fluorohydrocortisone. A careful history and physical exam is imperative in differentiating this subgroup of patients from classical Meniere's disease cases.

SALT LOAD ELECTROCOCHLEOGRAPHY

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Meniere's disease is a capricious disorder of the inner ear which may present either with its full quadrad of symptoms or in a more evolutionary manner with a single symptom manifesting months to years prior to the others. This makes the early diagnosis of Meniere's disease difficult and, when the initial symptom is vertigo, not only is the diagnosis difficult but localizing the condition to one or both ears is almost impossible. Electrocochleography has been helpful in this process but is insensitive during inactive phases. In an effort to increase electrocochleography sensitivity, salt loading patients presenting with symptoms compatible with Meniere's disease prior to testing is proposed.

To test the efficacy of this proposal, 20 normal volunteers will have baseline pure tone audiometry and extra tympanic SP/AP recorded. TIPtrode® earplugs will serve as the active electrodes. Alternating click stimuli will be presented at 95 decibels hearing level and 9.7 clicks per second. This study will then be repeated following "salt load" (4 grams sodium chloride/day for 3 days). The identical "salt load" protocol will be used on 40 patients with the symptoms of Meniere's disease but normal routine SP/AP ratios at baseline.

Preliminary results show that none of the ears from normal patients tested to date converted to abnormal ($SP/AP > .37$) while approximately 25% of the patients to date with normal pre "salt load" electrocochleography and the symptoms of Meniere's disease converted to abnormal in one or both ears. Based on these preliminary findings, it appears that "salt loading" a patient prior to obtaining electrocochleography recordings might improve the sensitivity of this test.

**LOW DOSE METHOTREXATE FOR THE TREATMENT OF
BILATERAL MENIERE'S DISEASE**

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Objective: To determine the effectiveness of long-term low dose methotrexate for the treatment of bilateral Meniere's disease refractory to traditional medical therapies.

Methods: The records from sixteen (16) patients with bilateral Meniere's disease of suspected autoimmune origin were reviewed retrospectively. All patients had chronic symptoms refractory to traditional medical therapy. Radiologic and metabolic workup revealed no other discernible etiology for their disease. All had a positive response to 2-4 weeks of oral prednisone (1mg/kg/day) before administration of treatment. All patients were treated for a minimum of one year with 7.5-15 mg/week of oral methotrexate and followed for a minimum of one year after treatment. Vertigo and hearing loss were evaluated before and after treatment.

Results: All patients completed treatment following the above guidelines. Fourteen (87.5%) patients experienced an improvement or complete resolution of their vertigo, and 2 (12.5%) had no improvement. Hearing improvement was defined as an improvement in pure tone threshold average greater than 10 dB or an increase in speech discrimination of greater than 12%. Hearing improved in 9 (56.3%) patients, was unchanged in 3 (18.7%) patients, and worsened in 4 (25.0%) patients.

Conclusion: Our findings suggest that some patients with bilateral Meniere's disease may have an (auto)immune-mediated component that is refractory to traditional medical management. Long-term administration of methotrexate at a low dosage comparable to that used for benign rheumatologic disease is a safe and effective therapy for the treatment of Meniere's disease in these patients.

THE USE OF MIDDLE EAR SUSTAINED RELEASE VEHICLES TO MORE APPROPRIATELY TARGET INNER EAR DISEASE

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Transtympanic gentamicin therapy has become a popular treatment for vertigo associated with Meniere's disease. Despite the increasing use of this modality a number of questions remain unanswered. The appropriate total dose, dosing frequency and the optimum end-point of therapy have not been established. More importantly little is understood about the basic properties of gentamicin when administered transtympanically. To help address these issues we have been investigating a number of sustained release devices. These devices allow us to control for many of the variables that are present in simple transtympanic administration. The device under investigation is placed in the middle ear of *Chinchilla laniger*. At set time points we sample the perilymph of the animal to determine gentamicin level and fix the inner ear for morphological analysis. Functional hearing assessment is performed with evoked potentials. Using a variety of different devices we have constructed inner ear kinetics curves which are specific to the device and drug dose. By correlating these curves with animal function and inner ear damage patterns we have learned a great deal about the basic properties of gentamicin. These findings have immediate implications in our patients. Since many of these devices are available for use in humans, it is important that physicians understand the properties of the devices. As we move beyond gentamicin and begin to use medicines to cure rather than simply treat inner ear diseases a basic understanding of the different classes of sustained release devices and the properties of the devices will become essential.

SELECTIVE LABYRINTHECTOMY IN EXPERIMENTAL ENDOLYMPHATIC HYDROPS*

Paul S. Bennett, M.D., Patrick J. Antonelli, M.D., Melanie Adamczyk, M.D.

Selective labyrinthectomy (SL) techniques allow for hearing preservation in the treatment of BPPV, but its feasibility in ears with endolymphatic hydrops is unknown. In this study, the guinea pig model was used to assess the cochlear effects of SL in newly-induced and chronic hydrops. Animals were randomized to undergo a hydrops procedure with 1) sham single canal ablation (SCA) after ten days, 2) SCA after ten days, 3) SCA after four months, or 4) sham hydrops procedure with SCA after ten days. Groups one, two and three showed gradual increases in auditory thresholds over the study period similar to control ears. Group four (late hydrops) showed a significant elevation in thresholds after canal ablation. These findings suggest that SL may be performed early in the course of hydrops with reliable hearing preservation, but SL in chronically hydropic ears is more likely to result in significant hearing loss.

* Funded by the Deafness Research Foundation

**OTOTOXICITY RESULTING FROM COMBINED ADMINISTRATION
OF METRONIDAZOLE AND GENTAMICIN**

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M. Rita Young, Ph.D., Timothy C. Hain, M.D., Gregory J. Matz, M.D.

The hypothesis that metronidazole can augment the ototoxicity of gentamicin was tested. Eight groups of five guinea pigs were given either a varying dose of gentamicin in combination with metronidazole, a varying dose of gentamicin alone, or metronidazole alone. Auditory damage was determined electrophysiologically by measuring the compound action potential and the alternating current cochlear potential. Hair cell damage was determined histopathologically by immunofluorescent preparation. An augmented ototoxic effect occurred when metronidazole was given with a moderate dose of gentamicin. Synergistic ototoxicity occurred in the apical and third cochlear turns when metronidazole was given with a high dose of gentamicin.

RECOVERY FROM AMINOGLYCOSIDE OTOTOXICITY???*

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250 patients administered aminoglycosides for treatment of life-threatening infections were tested for horizontal vestibular ocular reflex (HVOR) function using pseudorandom sinusoidal rotational stimuli. Of these 250, 20 were clinically normal at baseline testing, had no known prior history of vestibular/cochlear disease, and were followed for at least one year after the administration of the drug/s. Results were compared to an age-matched normal population and an age-matched population of hospitalized patients who did not receive aminoglycosides. The change in the HVOR of the amino patients could be characterized in 1 of 3 ways: a) a significant drop in HVOR function, with incomplete recovery (relative to baseline function) after one year; b) a significant drop in HVOR function with no recovery at one year; or c) no change in HVOR function. These changes (or lack of changes) were independent of cumulative dosage or type of aminoglycoside administered. These results suggest that complete loss of HVOR function caused by aminoglycoside ototoxicity is less common than transient loss of HVOR function. Caution needs to be taken however, as similar, transient losses were seen in some of the hospitalized control patients. The time course of recovery for both the aminoglycoside and the bed-ridden patients is slower than the recovery of the gain and phase of HVOR function seen in patients who compensate after unilateral vestibular neurectomy.

*Supported in part by NIH grants R01-NS19221 and R01-DC00204, and NASA grant NAGW-379

INTRACOCHELEAR PERFUSION WITH NO-DONATORS AND NOS-INHIBITORS IN GUINEA PIGS

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Introduction: Nitric oxide (NO) is synthesized by three different isoenzymes of NO-synthase (NOS I-III). Immunoreactivity for neuronal-type NOS I and endothelial type NOS III has been demonstrated in the cochlea of the guinea pig. NOS I immunoreactivity was seen in inner and outer hair cells, spiral ganglion cells, basal and intermediate cells of the stria vascularis, spiral ligament cells, and the media of vessels near the modiolus. An antibody to NOS III stained primarily vascular endothelial cells and less intense certain ganglion cells. Method: In the present experiments we tested the effects of the NO-donator S-nitroso-N-acetylpenicillamine, and the NOS-inhibitors N-nitro-L-arginine and N-nitro-L-arginine-methylester on sound-evoked responses of the cochlea. They were applied in different concentrations by intracochlear perfusions.

Discussion: The expression pattern of NOS in the cochlea is suggestive of various potential functions of NO in the inner ear. One could be the regulation of intracellular Ca^{2+} concentrations in the inner and outer hair cells which could influence both the mechanical properties of the hair cells as well as neurotransmission at synapses of the auditory nerve. Unimpaired blood supply is of major importance for cochlear function. NO is a vasodilator and inhibition of NOS could specifically decrease cochlear blood supply. The results of cochlear perfusion with NO-donator and NOS-inhibitor will be presented and discussed.

THE EFFECTS OF STRESS-RELATED HORMONES ON INNER EAR FLUID HOMEOSTASIS AND FUNCTION

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The inner ear maintains a delicate homeostasis necessary for proper auditory and vestibular function. Homeostasis disturbances is thought to cause certain diseases such as Meniere's disease. The pathophysiology of Meniere's disease is not completely understood. The discovery of endolymphatic hydrops in temporal bones of Meniere's patients and development of an animal model have enhanced understanding of the pathophysiology of this disease; however, the mechanisms leading to disturbance of inner ear homeostasis have not been elucidated. Several factors, such as stress-related hormones, may be involved in disruption of this delicate balance. Perilymph osmolality changes and functional disturbances have been reported following systemic epinephrine infusion.

This study investigated short and long-term effects of epinephrine administration on electrolyte concentration of perilymph and auditory function. Preliminary studies showed elevations in perilymph sodium and potassium after systemic infusion of epinephrine (6.3 ug/min for 3 hrs). Administration of epinephrine (500 ug/kg/day) for 30 days using an Alzet osmotic pump resulted in a 30 dB ABR threshold shift. Other biochemical changes in perilymph after long-term epinephrine administration will also be presented.

There is good evidence to suggest that stress-related hormones such a epinephrine can alter inner ear fluid homeostasis and auditory function. The present study confirmed this hypothesis and illuminated the process of alteration by demonstrating specific changes in perilymph composition and auditory function following acute and chronic epinephrine administration. These studies provide a stronger basis for further research to clarify the mechanisms of inner ear disturbances that lead to disease states such as Meniere's disease.

**BIOCHEMICAL MARKERS FOR IDENTIFICATION
OF HUMAN PERILYMPH**

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Perilymph fistula remains a controversial diagnosis because of the inability to objectively document the nature of fluid identified during surgical exploration of the middle ear. While beta-2 transferrin assays have successfully confirmed cerebrospinal fluid leaks, these have not been reliable when used to identify perilymph. Less sensitive assays such as those previously reported have a high false negative rate, primarily due to the limited and highly variable enrichment of beta-2 transferrin in perilymph relative to CSF. Additionally, an exquisitely sensitive assay utilized in the current study frequently detected trace amounts of this protein in serum, and could produce an unacceptably high false positive rate in clinical use. An alternative potential protein marker for human perilymph was detected and found to be highly enriched (70X) in human perilymph relative to serum. Purification and characterization methods identified this protein as human apolipoprotein D, and a sensitive chemiluminescent Western blot assay for this protein was developed. Samples that contained either microliter amounts of perilymph or random middle ear fluids were collected during inner ear surgery and tested blindly. Using the current assay system, the blinded investigator positively identified 15 of 20 perilymph samples (75%), with no false positive results among 15 negative controls. Contamination with blood may account for the five false negative results. Assays for apolipoprotein D show promise for assisting in the clinical diagnosis of perilymph fistula.

**BETA-2 TRANSFERRIN ASSAY IN THE
IDENTIFICATION OF PERILYMPH**

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BACKGROUND: Considerable controversy exists regarding the diagnosis of perilymph fistula. Recent studies suggest that detection of BETA-2 transferrin protein may be useful in the identification of perilymph. Investigations using this protein assay for samples collected during middle ear explorations in children with suspected perilymph fistulas have shown some positive results.

METHODS: In an effort to confirm the findings of previous studies regarding the utility of the BETA-2 transferrin assay for identifying perilymph, paired perilymph samples and negative controls were collected on gelfoam pledgets from 20 patients undergoing surgery which opened the inner ear. Immuno-electrophoretic assay for BETA-2 transferrin was performed on each specimen in a blinded fashion.

RESULTS: Only 1 (5%) of the known perilymph samples and 0 (0%) of the control specimens were definitely positive for BETA-2 transferrin.

CONCLUSIONS: These findings suggest that BETA-2 transferrin protein may not be a reliable marker for perilymph when performed using this collection and assay method.

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1998

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